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TOXOPLASMOSIS IN DOMESTIC ANIMALS:
Abortion and Stillbirth in Asymptomatic Carrier Gilts[1]

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Toxoplasma gondii is known to be pathogenic to human beings and many species of animals, including swine; however, toxoplasmosis on Taiwan has not yet been reported.

Since the demonstration of *Toxoplasma* in swine by Farrell *et al* (1) in 1952, attention has been paid to the role of swine as a reservoir of this protozoon (2-11). Sanger and Cole (12) suggested the possible transmission *via* uterus and milk in experimentally infected sows. The work of Nobuto *et al* (13) in 1960 indicated toxoplasmosis as a possible cause of abortion and stillbirth in swine. However, actual isolation and identification of *Toxoplasma* from naturally aborted or stillborn fetuses of swine was not reported until 1961 by Ogihara *et al* (14).

This report deals with the isolation and identification of *Toxoplasma* in swine for the first time on Taiwan where a serious outbreak of abortion and stillbirth occurred in the pregnant gilts on a local farm from January to May 1961.

MATERIALS AND METHODS

Tissue samples, including brain, were fixed in 10% aqueous formalin. These were embedded in paraffine and cut in 6μ thick, and stained with hematoxylin and eosin. Giemsa and Gram's stain were used when necessary.

Groups of 5-10 *dd* mice were inoculated intracerebrally (0.03 ml), or intraperitoneally (0.8 ml) with 10% tissue suspension in saline for isolation of Japanese B encephalitis virus, *Brucella* and *Toxoplasma*. Korthoph's medium was used for *Leptospira* isolation.

The following methods were used for serological testing. *Brucella*: plate agglutination test; *Leptospira pomona:* agglutination-lysis test (15); *Toxoplasma:* modified dye-test of Sabin-Feldman (16); Viral influenza: hemagglutination-inhibition test (17) for parainfluenza 1 (Sendai virus), and complement fixation test for influenza A2; Japanese B encephalitis: hemagglutination-inhibition test (18).

A *Toxoplasma* strain of swine origin, obtained from the National Institute of Animal Health of Japan, was used for control study. The antigen used for brucellosis was *Brucella abortus,* and was prepared by the Taiwan Provincial Institute of Animal Health.

The serological tests on *Brucella, Toxoplasma* and *Leptospira* were done in this laboratory, while those on viral influenza (including swine influenza), parainfluenza 1 (Sendai virus) and Japanese B encephalitis were kindly performed by staff of the Naval Medical Research Unit II, U. S. Navy, Taipei.

RESULTS

History of Abortion and Stillbirth in Pregnant Gilts

The pregnant gilts affected were breeds of Hampshire, Berkshire and Yorkshire. All were imported from Iowa, U. S. A., when they were 3-4 months of age and were proved to be *Brucella* free before shipping. All gilts had been vaccinated with attenuated hog cholera vaccine before breeding, and bred by natural service. None of the boars showed any abnormalities of testicles. A previous influenza-like epidemic was experienced in all herds of animals during December 1960 through January 1961. Sulfa drugs seemed to be

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effective in controlling this unknown malady; however, 7 gilts which had high fever aborted at that time. Abortion and stillbirth were then found in 51 out of 66 pregnant gilts (77%) from January to March 1961. Only 7 of them aborted with high fever, the other 44 gilts either aborted or delivered stillborn pigs without noticeable clinical manifestations. Most of the fetuses were stillborn, some mummified, and the few live ones either died within a few minutes or a few days after birth, or developed blindness or incoordination of lower limbs later.

It is worthwhile to know the relationship between the length of gestation and the status of abnormal farrowing in these herds, because such information might help in differential diagnosis. It is recognized that the length of gestation in swine is variable depending upon the breed; for the above purpose, however, the affected gilts are nevertheless classified arbitrarily into 4 categories according to the length of gestation: Early pregnancy (1 to 40 days), mid-pregnancy (41 to 80 days), late pregnancy (81 to 119 days) and delayed delivery (121 to 136 days), as shown in Table I. From the table, it is apparent that about 77.3% (34/44) of gilts farrowed either within the stage of late pregnancy or delayed delivery. As to the fetuses, the aborted, stillborn and mummified ones amounted to about 81.8% (219/268) of the total number of fetuses born while 49 young (18.3%) survived.

The 6 gilts which aborted and farrowed stillborn pigs in March were bred again during April-May, and they gave birth to 50 healthy youngs on the expected delivery date. The rest of the gilts were bred again in October and November, and gave birth at full term without any abnormalities.

Pathology

Fetuses. Five stillborn fetuses farrowed in late pregnancy from 3 individual gilts were examined 36 hours after delivery. Although these were kept in ice-jars during transportation, they were nearly decomposed upon arrival. 2 of these fetuses were covered by placentas. Since the pathological findings observed in these fetuses were similar, only general description will be made to avoid duplication. Although all visceras were examined, the following description includes only these visceras which showed definite pathological changes.

Lung. Microscopically, three fetuses had typical fetal lungs, while the other 2 had moderately expanded ones. Occasional tiny foci of coagulation necrosis were found here and there in the parenchyma. Many tiny, round or ovoid form organisms, with intensely stained blue nuclei and faintly stained red cytoplasm, were found in or close to the necrotic tissue. They stained blue with Giemsa stain, and were Gram positive. Occasionally, alveolar lining cells containing groups of 3-6 organisms in the cytoplasm were noted. Inflammatory cell infiltration was not found.

Heart. Grossly visible change was not found other than occasional tiny grayish foci on the epicardium. Microscopically, focal necrosis of cardiac muscle fibers in which great numbers of *Toxoplasma*-like organisms existed were frequently found (Fig. 1). The organisms were frequently seen in clumps encysted by poorly defined membranes in the otherwise intact muscle fibers at the vicinity of the necrotic foci (Fig. 2), or in isolated conditions without any noticeable host cell damage or cellular reaction around them. Moderate mononuclear infiltration was seen in the necrotic foci, subendocardial and subepicardial layers, and adipose tissue, where some of the macrophages contained a few organisms.

Liver. Microscopically, they were typical fetal livers in which active hemopoiesis was

TABLE I

The length of gestation of affected gilts and the status of abnormally farrowed fetuses

		Early pregnancy 1-40 days	Mid-pregnancy 41-80 days	Late pregnancy 81-119 days	Delayed delivery 120-136 days	Total
Affected gilts		3 (6.9%)	7 (15.9%)	30 (68.1%)	4 (9.1%)	44 (100%)
Fetuses	Abortion or stillbirth	?	7 (2.6%)	107 (39.9%)	14 (5.2%)	268 (100%)
	Mummified	0	6 (2.2%)	66 (24.7%)	19 (7.1%)	
	Survived	0	0	49 (18.3%)	0	

evidenced. Two livers were markedly autolysed; however, necrotic foci were still demonstrable. Many tiny foci of coagulation necrosis were found in the acini (Fig. 3). *Toxoplasma*-like organisms were found in the cytoplasm of hepatic cells and Kupffer cells, within or close to the necrotic foci.

Lymph nodes. Typical follicular formation was not found, and lymphocytic cells grew diffusely. Diffusely necrotic lymphocytic cells were found in the cell poor substance of the pulmonary lymph nodes. Occasionally, extra-cellular organisms were found in the necrotic area.

Central nervous system. Three brains were autolysed. In the other two cases, no grossly visible changes were found. Microscopically, the brains and spinal cords were equally affected. There were many glial nodules, mainly composed of microglias, scattering in the gray and white substances. Active neuronophagia was found everywhere. Large *Toxoplasma* cysts were noted in both brains but particularly abundant in one case. They were usually seen at the margin of the glial nodules (Fig. 4 & 5). Occasionally, a few extra-cellular organisms were found in glial nodules also. No *Toxoplasma*-like organisms were found in the spinal cord. Perivascular cuffing was meager.

Placenta. The 2 placentas examined were nearly decomposed. In one placenta many small, flat, whitish-gray, hard foci were seen here and there. Microscopically, intensively calcified foci scattered in the mesothelial layer of chorionic membrane; moderate calcification was also noted in the mesothelial layer of villi. Trophoblasts remained intact. No *Toxoplasma*-like organisms were demonstrable in the placenta.

The other placenta showed slight calcification in the mesothelial layer of the chorionic villi. Necrotic or necrobiotic trophoblasts desquamated into the outer space, in which bacterial clumps were frequently noted.

Case 60-85. This pig was a well nourished male Yorkshire, 2 months of age. All its littermates were stillborn, and this was the only one that survived. It showed pneumonic signs for about 1 month, and was found to be blind. On gross examination no corneal lesions were found, only slightly opaque pupils were noted. Bronchial rale was noted at the right anterior region of the chest. A blood sample was collected right before it was killed, and the dye-test titer for *Toxoplasma* was found to be 1:4096. The dam of this pig gave antibody titer of 1:256 when it gave birth in May.

Integument. Moderate edema was found in the subcutis of the neck. Microscopically, diffused edema and sparsely infiltrated neutrophils were seen in the subcutis.

Lung. A consolidated area about 2×3×3 cm in size was seen in the outer margin of the right middle lobe. A reddish consolidated area measuring 2×4×3 cm was seen in the cardiac lobe. A small amount of cloudy mucus was found in the bronchi. Microscopically, atelectasis was frequently seen at those portions where no gross lesions were apparent. Thickened alveolar walls in grossly consolidated areas were caused by slightly hyperplastic alveolar lining cells and slight mononuclear infiltration. In some lobules, one or two alveoli collapsed focally to give the thickened appearance of the alveolar walls.

In many lobules, cuboidal hyperplastic alveolar lining cells were arranged in the form of glandular ducts (Fig. 6). Bronchiolar epithelial cells and alveolar ducts were hyperplastic and taller than normal. Marked reticular fiber proliferation was noted around the arteries. Increased fibroblastic element was also found in the peribronchiolar interstitial tissue. Occasional fibrosis of alveolar walls was also noted.

Marked hyperemia, edema and moderate neutrophilic infiltration were seen in the alveoli of some lobules; capillaries of the alveolar walls were markedly hyperemic. Edema in interlobular connective tissue was obvious in some locations.

Heart. Mononuclear aggregated foci were often found which contained some necrotic myocardial fibers. *Toxoplasma* in groups was found in some myocardial fibers without any cellular damage or cellular reaction.

Brain. A few glial nodules were found.

Liver. Tiny granulomatous foci were occasionally found in the acini. Mild mononuclear infiltration was seen in the portal area.

Kidney. A few tiny whitish spots were seen in the cortex beneath the capsule. Microscopically, focal mononuclear infiltration in the interstitial area was frequently noted here and there in the cortex; a few glomeruli and renal tubules were missing.

Eye ball. Opaque pupils were noted without corneal involvement. Microscopically, marked mononuclear infiltration was seen in the markedly disorganized retina and in the choroid membrane (Fig. 7). Marked hyperemia, slight hemorrhage and slight lymphocytic infiltration with *Toxoplasma*-like organisms scattered in the stroma were seen in the ciliary ring. Occasionally large glial nodules were seen in the optic nerves (Fig. 8). Granulomatous foci and moderate mononuclear infiltration were frequently found in the degenerative and necrotic muscular bundles or in the intermuscular connective tissue (Fig. 9); giant cells or multinucleated mucular fibers were also noted occasionally in these foci.

Skeletal muscles. Mononuclear infiltration in the necrotic muscular fibers and intermuscular connective tissue was frequently observed.

Microbiology and Serology

Microbiology. *Brucella* organisms were not isolated from the gastric contents, spleen, and liver of 5 stillborn fetuses. The rapid agglutination test, using *Brucella abortus* as the antigen, was negative at 1:50 on all serum samples collected from 12 affected gilts.

Attempts of *Leptospira* isolation from 4 stillborn fetuses and serological test for *Leptospira pomona* on 34 serum samples collected from affected gilts also yielded negative results.

Those mice which were inoculated intraperitoneally with fetal brain emulsions did not show any abnormal signs during the 21-day observation period. The second passage with brain of mice through the same route also gave negative results. The 10 mice which were inoculated intraperitoneally with the lung emulsion prepared from Case 60-85 became dull and sluggish in movement at the 12th day after inoculation. A large amount of viscid, semitransparent ascites, in which a small amount of fibrin floated, was drawn from mice by paracentesis, and fresh and smear preparations were made therewith. Abundant microbes mixed with large numbers of mononuclear cells were found in the fresh preparation. These micro-organisms were pleomorphic, but mostly lunate or pyriform in outline; some of them manifested a sluggish, undulating movement throughout the entire body. Giemsa stained smears revealed organisms with similar morphologic features (Fig. 10), with a size 2-3.6 $\times$4-7.5μ. A solidly-stained nucleus was usually located close by the rounded end of the organisms and sometimes it was in a central position. The morphological characteristics described above agree with the classical description of *Toxoplasma* in literature, and they also match very well with a swine strain of *Toxoplasma* maintained in this laboratory. Identification of this unknown microbe as *Toxoplasma* was further established by the modified dye-test of Sabin and Feldman (16): the same end-titer of 1:4096 was attained on serum of pig 60-85 from which the organisms under study had been isolated, by both antigens of the known *Toxoplasma* strain and the unknown organisms under test. Normal serum and physiologic saline controls were negative for cytoplasm-modification.

Pathogenicity of the organism isolated was tested on 4 healthy pigs, each weighing about 20 kg, whose serum contained no demonstrable cytoplasm-modifying antibodies. Two pigs (61-38 and 61-39) were inoculated intratracheally with 5 ml of diluted mouse ascites which contained 6.5 million microbes, and another 2 non-inoculated pigs were placed in the same pen to see if contact infection would occur. Both of the inoculated pigs showed elevated body temperature on the 3rd day after inoculation. The high fever in pig 61-38 continued for 10 days, ranging 40-41.8 C, before dropping to normal value. Dyspnea and non-productive coughing were observed during the 21-day observation period. The high fever in pig 61-39 continued for 5 days, ranging 40.4-42.2 C and it died on the 8th day after inoculation. *Toxoplasma* was observed in the smears prepared from obviously pneumonic lungs of pig 61-39. The body temperature of non-inoculated pigs raised at 6 and 7 days and dropped to normal level at 11 and 12 days respectively after experimentation. The fever did not exceed 41.0 C, and the animals recovered quickly therefrom.

Blood samples were drawn periodically from these pigs for antibody titration, and the results are shown in Table II. It is obvious from this table that pigs responded equally to experimental inoculation with isolated *Toxoplasma* as well as to contact infection. The overall findings described above indicate, therefore, that the protozoon in question was pathogenic for pigs.

Serology. Seven serum samples from affected gilts examined in May gave dye-test titers for *Toxoplasma* at least 1:64. Additional blood samples from 88 gilts, including 51 affected ones, and 10 boars were accordingly collected for antibody titration in June, and the result is shown in Table III. It shows that about 80% of the gilts and 40% of the boars of this group had antibody titers at least 1:16. Consecutive titration data as given in Table IV indicate that diagnostically significant titers persisted for quite a long period, because the last titration in August was done 5 months after the epizootic peak had subsided.

Abnormally farrowing gilts had significantly more positive reaction than normal farrowers (Table V). It will be noted that 46.6% of the latter group had serological evidence of *Toxoplasma* infection, and yet they farrowed normally.

Dye-test was performed on serum samples collected from 119 young pigs (7-11 months of age) which were born during the outbreak of the disease (Table VI). 28 of these pigs were born from affected gilts and the rest from normally farrowing ones. Table VI shows that 50% of the pigs from affected mothers, and 22.2%

TABLE II

Serological response of pigs experimentally infected with the isolated toxoplasma organism

Treatment	Pig no.	Days elapsed after inoculation						
		Pre-inc.	4	8	14	21	29	99
Intratracheal inoculation	61-38	<4*	4	4	16	16	4	4
	61-39	<4	4	4	(died)			
Contact	61-40	<4	<4	4	16	16	256	16
	61-41	4	4	4	4	16	64	16

* Reciprocal of serum dilution in Sabin-Feldman dye-test.

TABLE III

Dye-test on serum samples of all gilts and boars in a farm, collected in June 1961

	Titers						Total
	Negative	Positive					
	≦4	16	64	256	1024	4096	
Gilts	18	17	22	19	10	2	88
%	20.5%	79.5%					100%
Boars	6	2	0	1	0	1	10
%	60%	40%					100%

TABLE IV

Persistence of antibody titer in gilts with toxoplasmosis (modified dye-test of Sabin-Feldman)

Breed	Gilt no.	Serum collection time		
		May	June	August
Yorkshire	13		256	64
	31		1024	256
	98	256*	256	
Hampshire	139	256	256	
	669	64	64	
	699	256	64	256
	900		256	256
	909		256	64
	910	64	64	
	935		256	1024
	936		256	256
	9000		256	64
	9903		256	256
	9906	64	64	
	9953		1024	256
Berkshire	41		256	64
	45	1024	1024	256
	46		256	256
	49		256	256
	72		256	256
	73		1024	16384
	76		1024	1024
	953		256	256
	3021		4096	256
	3026		256	64

* Reciprocal of serum dilution giving positive reaction.

TABLE V

Results of dye-test on serum samples of pregnant gilts collected in June 1961

Breed	Normal farrower		Abnormal farrower	
	Negative*	Postive**	Negative*	Positive**
Yorkshire	1	2	3	10
Hampshire	4	4	2	24
Berkshire	3	1	0	12
Total	8	7	5	46
%	55.4	46.7	6.8	90.2

* Negative dye-test with 1:4 serum dilution.

** Positive dye-test with at least 1:16 serum dilution.

TABLE VI

Dye-test titer of pigs born on the farm during the outbreak of toxoplasmosis*

Gilts \ Young*		Antibody titer	
		4	>16
Normal farrower	Serologically positive (6 gilts)	$^{34}/_{40}$ (85%)	$^{6}/_{40}$ (15%)
	Serologically negative (11 gilts)	$^{37}/_{51}$ (72.5%)	$^{14}/_{51}$ (27.5%)
Abnormal farrower (9 gilts)		$^{14}/_{28}$ (50%)	$^{14}/_{28}$ (50%)

* Bled for antibody titration at 7-11 months of age.

(20/91) of the pigs from apparently normal mothers, gave positive titers of 1:16 or above. Furthermore, the test was positive in 27.5% (14/51) of the pigs born from serologically negative gilts.

Random serum samples of 29 gilts were tested for antibodies of viral influenza and Japanese encephalitis. Serological data given in Table VII indicate that none of these swine had recent infections of influenza A viruses (including swine influenza) or parainfluenza 1 (Sendai virus). Contrarily, hemagglutination-inhibition test with Japanese encephalitis virus antigen was invariably positive when sufficient serum was available for testing.

DISCUSSION

Since the criterion of diagnostically significant titer for *Toxoplasma* dye-test in swine has not been established, the current practice of taking 1:16 as the diagnostically significant titer in men (16) was adopted tentatively in this study. This policy is probably valid in view of the results of experimental inoculation and contact infection obtained (Table II).

As shown in the text, the possible correlation of brucellosis and leptospirosis, both being the usual causes of abortion and stillbirth in swine, with the malady reported here is definitely excluded. Nor was viral influenza to be blamed, since serological testing for influenza antibodies in affected gilts was invariably negative. In our opinion the validity of *Toxoplasma* as the causative agent of abnormal farrowing reported here has been satisfactorily established, because of the following evidence. Firstly, rising of specific antibody titers for *Toxoplasma* has been demonstrated in consecutive serum samples obtained from affected gilts (Table IV). Secondly, organisms which proved to be typical *Toxoplasma* in aspects of morphology, serology and pathogenicity have been isolated from a diseased young.

The frequent occurrence of antibodies for Japanese B encephalitis in the herd under study is not surprising, because the local swine populations have been shown to be widely infected with this virus (19). This finding can be interpreted to represent, in all probability, a background of inapparent infection acquired by the gilts after importing. Attempts to isolate Japanese B encephalitis virus by intracerebral inocula-

TABLE VII

Serological study on random serum samples of gilts

Serum no.	Status of fetuses			Dye-test, *Toxoplasma*	HI test, Jap. encephalitis	CF test, influenza A2 (soluble antigen) J-305	HI test, para-influenza 1 (Sendai virus)
	Alive	Stillborn	Mummified				
Y 31	0	0	12	>1024	nd*	—	—
Y 98	3	3	2	256	40	—	—
Y 99	1	0	2	nd	nd	—	—
Y3037	8	0	1	nd	160	—	—
Y3043	7	0	2	4	nd	—	—
Y3044	6	0	0	4	nd	—	—
Y3049	6	0	0	64	20	—	—
H 152**				nd	160	—	—
H 153**				nd	80	—	—
H 166**				nd	nd	—	—
H 203**				nd	nd	—	—
H 699	0	4	3	64	nd	—	—
H 956	8	0	0	16	nd	—	—
H 906	0	6	0	64	nd	—	—
H 936	0	4	1	256	40	—	—
H 999	8	0	0	64	80	—	—
H9906	0	4	5	64	nd	—	—
B 76		aborted		1024	nd	—	—
B 215	7	0	0	<4	160	—	—
B 402	8	0	0	nd	nd	—	—
B 415**				nd	nd	—	—
B 943**				nd	nd	—	—
B 975**				nd	nd	—	—
B1016**				nd	40	—	—
B3022	1	3	3	64	160	—	—
B3026	0	6	0	256	20	—	—
B3028	7	1	4	16	80	—	—
B3043**				256	nd	—	—
B3046	0	8	0	1024	40	—	—

nd* not done due to insufficient serum for testing.
** non-pregnant gilts.

tion in mice with brain emulsions from stillborn fetuses yielded negative results, so this virus is unlikely concerned in the abnormal farrowing observed. In this connection we do not share the views expressed by some Japanese authors (20-27) that mummified fetuses are characteristic features of stillbirth in swine caused by Japanese B encephalitis virus. We believe that fetal mummification, a non-specific phenomenon observed in this study too, would occur whenever fetuses die in mid-pregnancy.

According to Jacobs *et al* (28), cysts of Beverley strain of *Toxoplasma* in mouse brain survived for as long as 68 days at 4 C. Despite our successful isolation of *Toxoplasma* from fresh materials, our effort to isolate this organism from the nearly decomposed dead fetuses proved futile. The failure is rendered all the more perplexing, due to the demonstration of proliferative form as well as cysts of *Toxoplasma* in the histological preparations. Probably morphologically intact organisms observed were not neccessarily viable ones, and the decomposition process in the dead fetuses somehow rendered the *Toxoplasma* organisms nonviable.

It is a point of interest in differential diagnosis that a time difference exists between toxoplasmosis and hog cholera as etiologic causes of stillbirth in swine. In toxoplasmosis as shown in the present study (Table I), the majority of stillbirths occurred at late pregnancy, *i.e.* 81-119 days of gestation. In the case of hog cholera on the other hand, delayed delivery of the dead fetus (*i.e.* later than 120 days of gestation) is the main feature of abnormality (29).

The fact that about 50% of offspings of affected gilts gave negative dye-test to indicate that not all the fetuses are necessarily targets of *Toxoplasma* infection in the uterus. On the other hand, all 4 litters of 29 pigs which showed a negative dye-test titer were normally farrowed from 4 positive gilts. It seems to indicate that the gilts were in the state of past infection, that *Toxoplasma* no longer parasitized in the gilts, and the positive dye-test titer does not always

necessarily mean a chronic infection. Our unpublished data on the experimentally infected pigs seem to support this finding: *Toxoplasma* was not isolated in mice, while the antibody titer of one of the experimental pigs remained ×16 when killed for examination 5 months after the artificial inoculation.

About 27.5% of those pigs which were born from non-affected gilts (Table VI), with a negative dye-test titer, were positive for a dye-test. All pigs were separated from sows, and were collected in a herd after weaning. Some of them had shown pneumonic signs. It seems to have been due to the fact that those pigs which showed a positive dye-test titer acquired an infection during this pooling period.

Experimental contact infection of toxoplasmosis in swine has been successfully accomplished in this study under conditions simulating natural living, but the transmission route remains obscure. Some information has also been obtained regarding the length of incubation period in toxoplasmosis of swine. Under the condition of our experiment, pigs developed the infection sign of elevated body temperature 3 days after pneumonic signs and fever appeared in the primarily infected animals. The incubation period of natural toxoplasmosis in swine may be not much more than 8 days therefore, providing that transmission factors are favourable.

Calcified foci in the brain and hydrocephalus were frequently described in congenital toxoplasmosis in human fetus, but none of our stillborn swine showed these lesions. Instead the chorion membrane of affected gilts showed a marked calcification. Evidently the placenta was the primary site of *Toxoplasma* invasion in pregnant gilts. In one case, trophoblasts remained intact while the mesothelial layer was markedly calcified; while in the other case, trophoblastic necrosis was found besides calcification of the mesothelial layer. Secondary infection was apparent in the latter case.

The blindness of pig 60-85 was caused by severe chorio-retinitis and damage in the optic nerves. Since uveitis frequently develops in individuals with relatively low although steady antibody levels, Remington *et al* (30) commented that spontaneous parasitemia may be expected under such conditions and lodgment of parasites in tissues with low antibody levels may be brought about by transmission through wandering cells. The case cited above had high antibody titer of 1:4096, which represented, very likely active antibody formation instead of passively transferred antibodies because the dam had a much lower titer of 1:256. It seems unlikely that chorioretinitis in this case developed in the presence of high concentration of circulating antibody, and alternatively, the latter might probably represent the result of immunological response of the young to active infection. However, it is not apparent whether this pig was affected prenatally or post-natally.

In this study, all gilts recovered from toxoplasmosis were able to farrow normally several months after the previous abnormal delivery. This finding conforms with former reports on toxoplasmosis in human being and animals (31-37). About 28% of the young gave a positive dye-test, but it is uncertain whether it represents residual antibodies passively transferred from the dams or active antibody production on the part of the baby pigs.

SUMMARY

Toxoplasma gondii was isolated and identified as the cause of a severe outbreak of abortion and stillbirth in a swine herd. This is the first report of toxoplasmosis on Taiwan.

The role *Toxoplasma* played in the abortion and stillbirth syndrome in pregnant gilts was discussed in aspects of etiology, symptomatology, pathology and immunology.

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Illustration of Plates:

Fig. 1. A focal necrosis in the myocardium. Stillborn fetus. H. & E. stain, 80×.

Fig. 2. Enlarged view of *Fig. 1.* Arrows show groups of *Toxoplasma* in myocardium. H. & E. stain, 320×.

Fig. 3. Focal coagulation necrosis of the fetal liver. H. & E. stain, 180×.

Fig. 4. A large glial nodule in the cerebral cortex. Arrows show two large *Toxoplasma* cysts at the periphery of the glial nodule. Fetal brain. H. & E. stain, 80×.

Fig. 5. Enlarged view of *Fig. 4,* two cysts are visible. H. & E. stain, 320×.

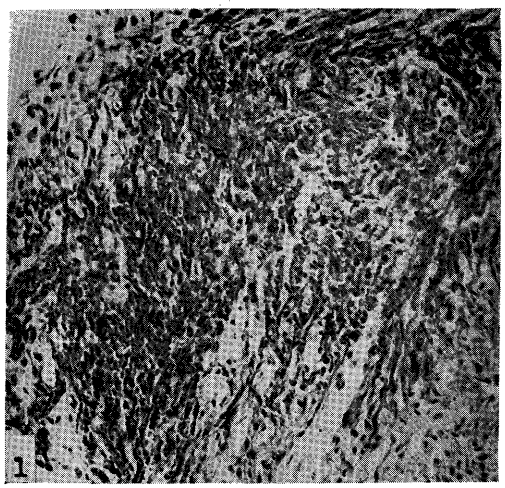
Fig. 6. Adenomatous arrangement of hyperplastic alveolar lining cells. Case 60-85. H. & E. stain, 320×.

Fig. 7. Marked mononuclear infiltration in the retina. The choroid membrane detached during the sectioning. Case 60-85. H. & E. stain, 200×.

Fig. 8. A glial nodule, mostly composed of microglias, seen in the optic nerve. Case 60-85. H. & E. stain, 200×.

Fig. 9. Marked mononuclear infiltration in the necrotic and fragmented muscular fibers. Case 60-85. H. & E. stain, 200×.

Fig. 10. Toxoplasma gondii isolated from a pneumonic lung of Case 60-85. Smear of the peritoneal exudate of an infected mouse. Giemsa stain, 1,000×.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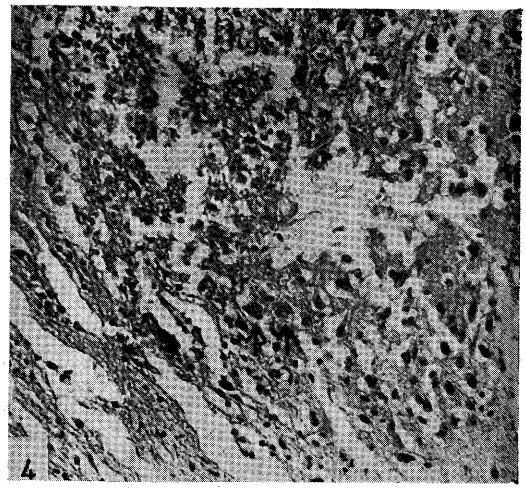
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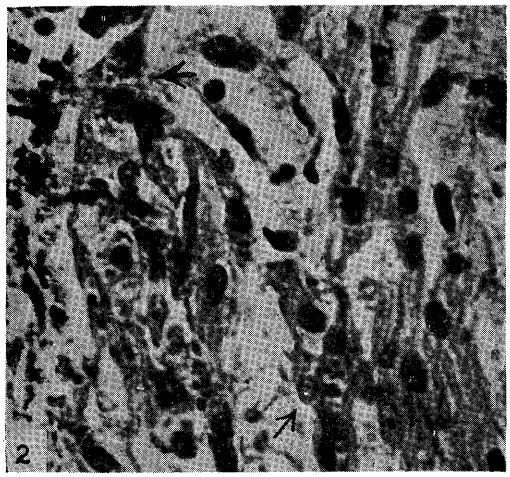
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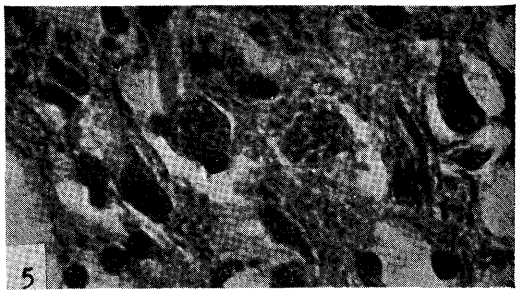
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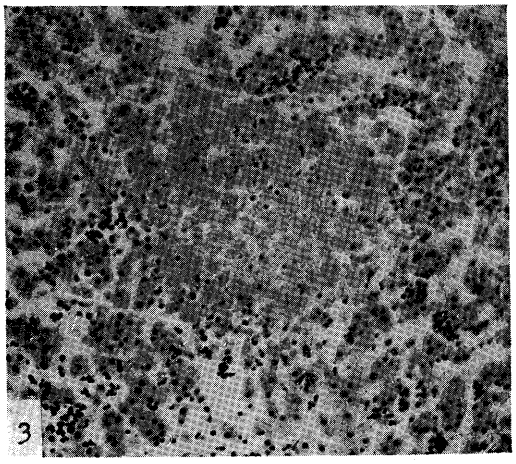
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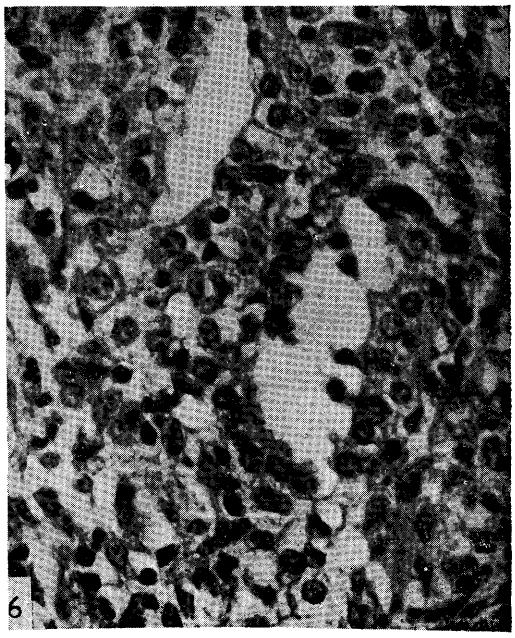
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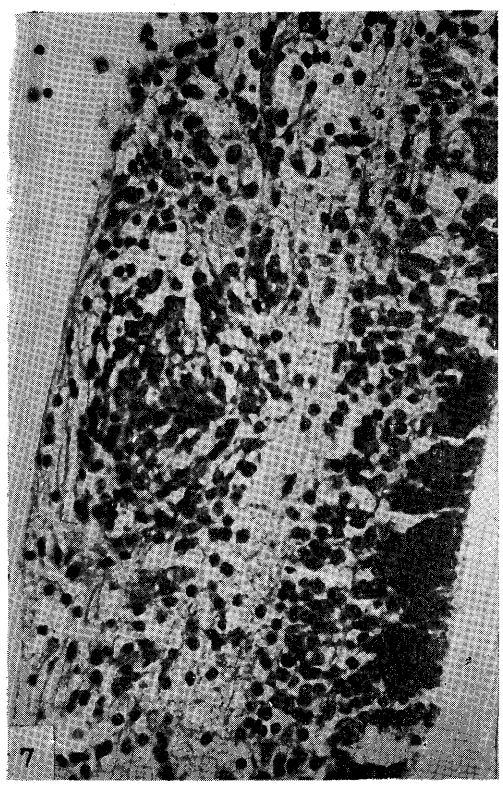

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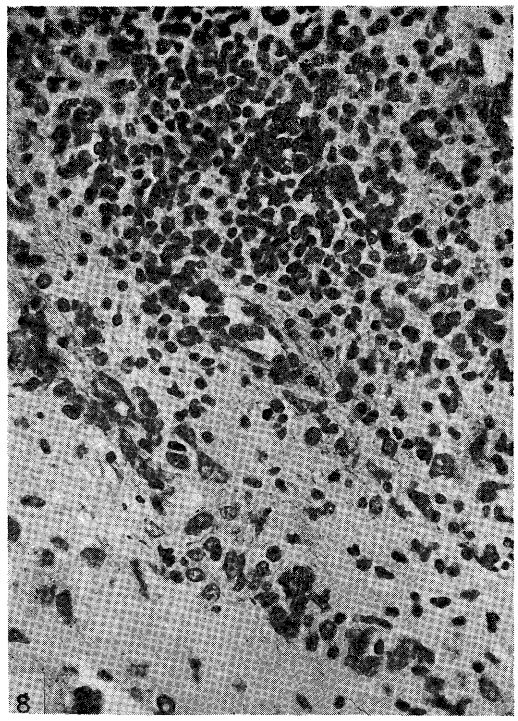

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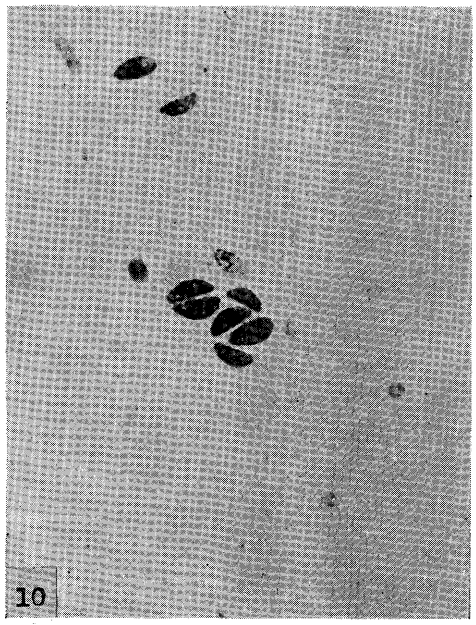

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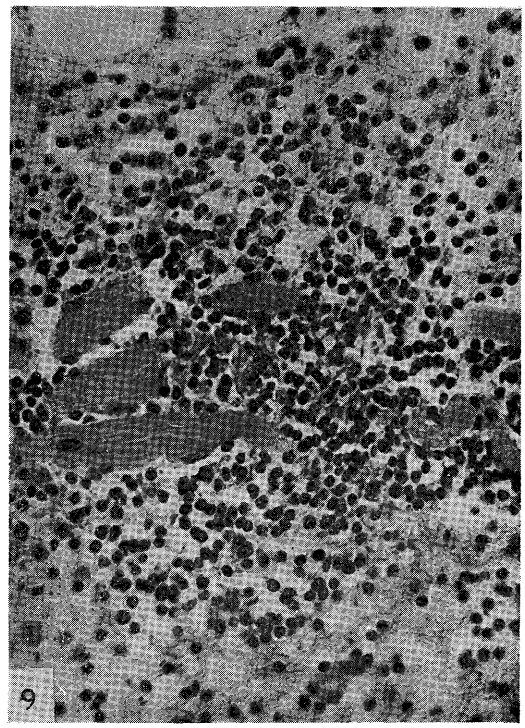

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